Respiratory Changes and Consequences for Treatment of Decompression Bubbles Following Severe Decompression Accidents

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INTRODUCTION

Earlier papers describe the theoretical and experimental work carried out to determine the best treatment strategy following severe decompression accidents during use of self-contained breathing apparatus such as the Canadian Underwater Minecountermeasure Apparatus (CUMA). The previous paper has described the extent to which decompression bubbles were formed in anaesthetised animals subjected to controlled primary and treatment hyperbaric procedures; the range of bubble counts was from zero to fatal. Treatment recompression apparently removed the bubbles quickly but in many cases this was not followed by an improvement in the condition of the animal and death occurred during the treatment. The experiments were designed to require minimal surgical intervention prior to the experiments to reduce the possibility that bubble numbers were influenced by surgery and indwelling catheters. There is therefore relatively little information available from which to draw conclusions about the immediate cause of death. Respiratory gases were monitored and a study of the information contained in those data sheds some light on this and allows a tentative conclusion to be drawn.

EXPERIMENTS

As described in the previous paper the experiments used either air or oxygen as the breathing gas during the 10 minutes surface interval between the end of the rapid primary decompression and the start of the treatment recompression. The experiments in which oxygen was breathed are the most useful for present purposes as all animals survived to start treatment including those with very high bubble counts. Five animals completed treatment. Table 1 summarises the outcome of these ten experiments.

Inspiratory and expiratory gas bags are used to enable use of controlled inspired gas mixtures and measurement of expired gases (Reinertsen et al 1998). Oxygen concentrations are measured in both. The volume of each bag is about 1 litre, equivalent to 5 or 6 tidal volumes, and therefore reflect gas switches fairly rapidly at normal respiratory rates. In addition a fast response carbon dioxide analyzer draws gas from the trachea so that end tidal carbon dioxide levels can be determined.

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Table 1
Maximum recorded bubble count in pulmonary artery and femoral vein

	PA Bubbles (/cm^2)	Femoral bubbles	Outcome
Expt 7	5.6 (94)	0	Completed USN6A
Expt 8	0.07 (99)	386	Completed USN6
Expt 2/1	20.52 (96)	1175	Died during USN6
Expt 2/2	0.04 (86)	0.146	Completed USN6
Expt 2/3	16.01 (86)	1964	Died after USN6A compression
Expt 2/4	22.1 (85)	1928	Died during USN6
Expt 2/5	21.07 (92)	1642	Died after USN6A compression
Expt 2/6	16.74 (94)	1694	Completed USN6
Expt 2/7	0.04 (86)	0	Completed USN6A
Expt 2/8	20.94 (89)	1825	Died after USN6A compression
Average	**12.3 ± 9.7**		

RESULTS

It is possible to derive some indication of respiratory function from the amount of oxygen extracted by the lungs, the inspired-expired oxygen partial pressure difference. This gives a crude measure of overall ventilation:perfusion balance though it is necessary to exercise some care in interpreting results in animals which have decompression bubbles. For example though a relative hypoventilation, ventilation lowered to an inappropriate extent compared to pulmonary blood flow, will give an increased inspired-expired oxygen difference; reduction in both ventilation and pulmonary blood flow can give a completely normal inspired-expired difference. An inspired-expired difference of zero can probably be safely taken as an indication that the animal is dead but the values can go from grossly abnormal to being apparently normal shortly before death as the failing circulation drops to a level which matches the reduced ventilation. An extremely low breathing rate results in a delay in the effect of a gas switch being transmitted to the expiratory bag which can result in negative values for the oxygen difference.

Table 2 shows oxygen difference before the primary compression and before the primary decompression for the experiments listed in Table 1, the animals breathing oxygen during the surface interval. Maximum bubble counts are also listed. Up to the start of the primary decompression all animals appeared to be similar, there is no indication of those animals which will have the highest bubble numbers.

Table 3 shows the same parameter:- at the end of the surface interval; on completion of the treatment compression; after a period of oxygen breathing at 18 metres; on completion of treatment. The five animals which survived to complete treatment did not have greatly changed oxygen differences after the primary decompression and returned almost to pre-experimental values after the treatment. Three had bubbles; #7 had 5.6 bubbles/cm^2 (equivalent to Doppler grade IV- according to Eftedal et al 1998), # 2/2 had 1.14 bubbles/cm^2 (Doppler grade III) and # 2/6 had 16.7 bubbles/cm$_2$. Experiment 2/6 is of particular interest as that level of bubbling proved fatal in other animals. With the exception of # 2/5 the animals which did not survive had abnormally high oxygen differences at some point during the experiment. Again animal 2/6 is different, being characterised by apparently normal oxygen extraction throughout despite the very high bubble count.

Table 2
Inspired-expired oxygen difference (kPa)

	Pre-compression	Pre-decompression	Bubble count
Expt 7	1.67	5.55	5.6
Expt 8	1.41	4.77	0.07
Expt 2/1	2.63	7.42	20.5
Expt 2/2	1.97	4.64	1.14
Expt 2/3	2.32	5.11	16.0
Expt 2/4	1.90	5.52	22.1
Expt 2/5	0.33	6.33	21.1
Expt 2/6	1.89	5.24	16.7
Expt 2/7	2.05	5.52	0.04
Expt 2/8	2.01	4.94	20.9
Mean ± St Dev	**1.82 ±0.62**	**5.50 ± 0.83**	

Table 3
Inspired-expired oxygen difference (kPa)

	Surface Interval	Post Treatment Compression	After oxygen breathing	After Treatment
Expt 7	6.58	1.85	1.75	1.64
Expt 8	2.80	2.51	3.24	2.01
Expt 2/1	11.94	122.0		
Expt 2/2	3.62	2.28	2.43	1.94
Expt 2/3*	27.0	-0.05		
Expt 2/4	4.99	107.1		
Expt 2/5*	2.92	-0.87		
Expt 2/6	2.82	3.54	3.88	2.52
Expt 2/7	3.96	2.64	2.53	2.08
Expt 2/8*	18.54	-0.13		
Mean ± St Dev			**2.77 ± 0.82**	**2.04 ± 0.32**

* animal dead at the end of treatment compression

DISCUSSION

Animal 2/6 survived the procedures against the odds. A bubble count of 16.7 /cm^2 would usually prove fatal. This animal's gross pulmonary function appeared not to be deranged by that extent of bubbling. A similar level of bubbling in experiment 2/3 led to early death following an abnormally high oxygen extraction. What is the difference? Were there any critical differences in the lungs of these animals? Should we be considering some kind of pulmonary therapy during treatment in this kind of accident?

There is a clue to the cause of the differences between these animals in the pattern of respiration. Tidal ventilation of the lungs can be seen on the trace which records end-tidal carbon dioxide concentration. For this purpose a useful comparison can be made between number 2/4 and 2/6 both of which had USN6 as treatment, 2/4 died 30 minutes after disappearance of the bubbles, well into the treatment. Figures 1 shows the breathing rate for experiment 4 together with the pressure profile and the bubble counts; the respiratory rate for experiment 6 is displayed as a box enclosing all values. The very low respiratory rate of animal 4 explains the high oxygen extraction; the rate dropped as bubble count increased but did not rise again as the bubbles were reduced during treatment. There appears to have been some kind of irreversible change in animal 4 which was not experienced by animal 6.

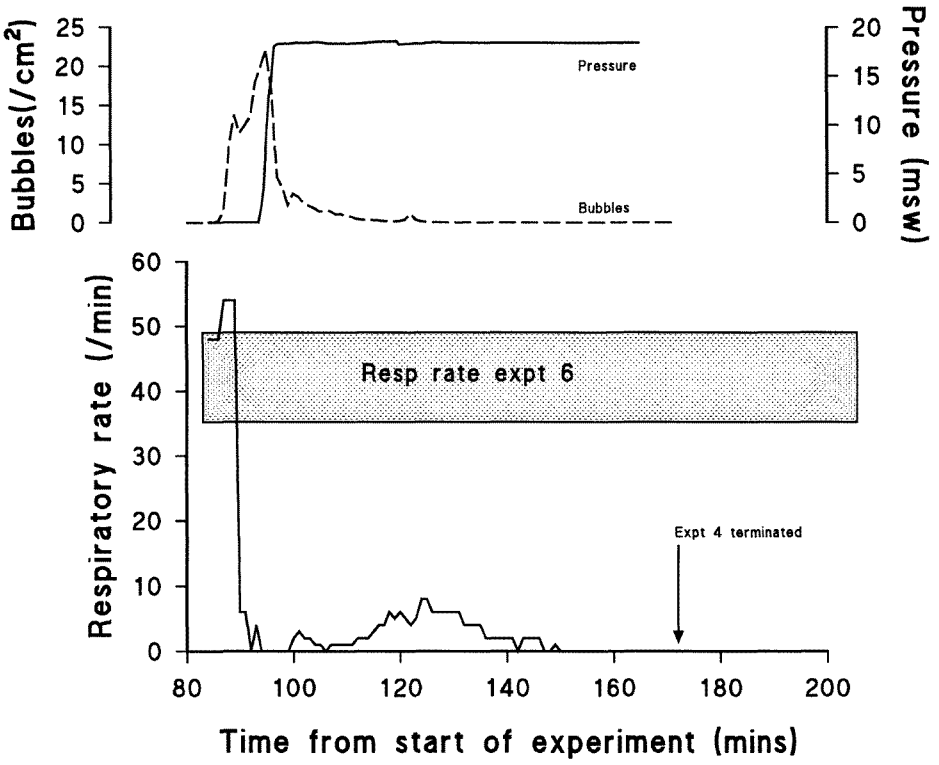
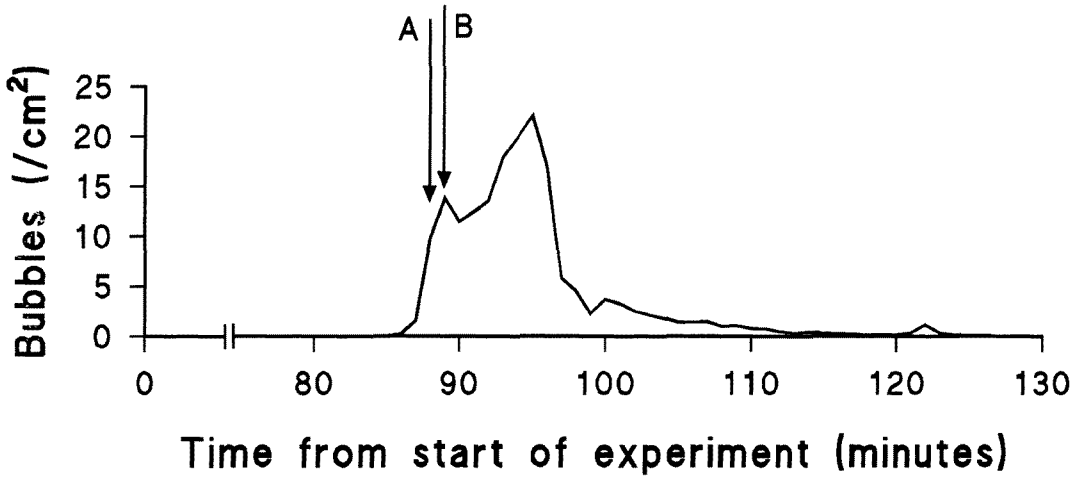


Figure 1 Respiratory rate, pressure profile and bubble counts for experiment 2/4.

Figure 2 shows pulmonary artery bubble counts and end tidal carbon dioxide levels for animal 4 over part of two consecutive minutes as bubbles developed. Figure 3 continues with end tidal carbon dioxide half a minute after the second trace in figure 2 and also 30 minutes later. These figures illustrate the complete abolition of a regular respiratory pattern within the space of about 2 minutes. The final respiratory pattern is prolonged expirations with irregular single inspiratory efforts, apnoea.



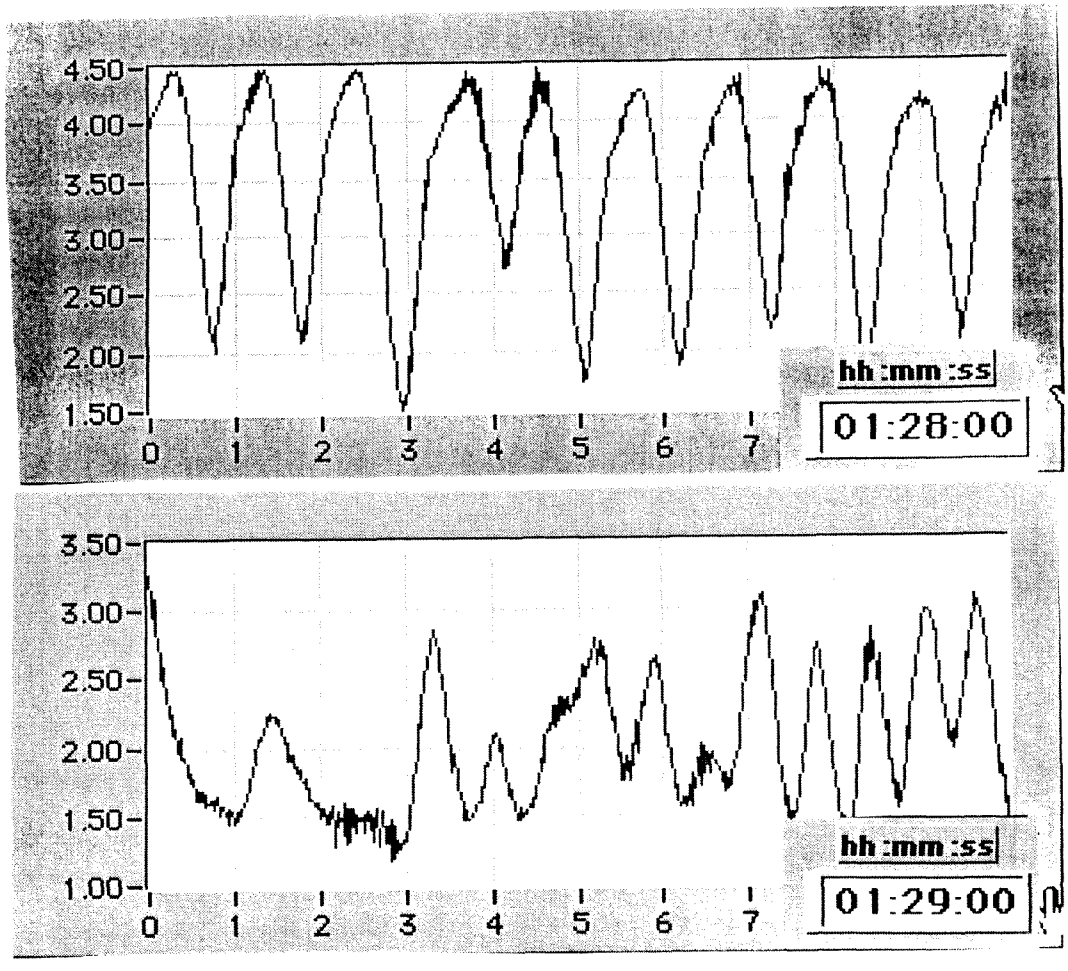


Figure 2 Respiratory patterns, experiment 2/4. See text for details

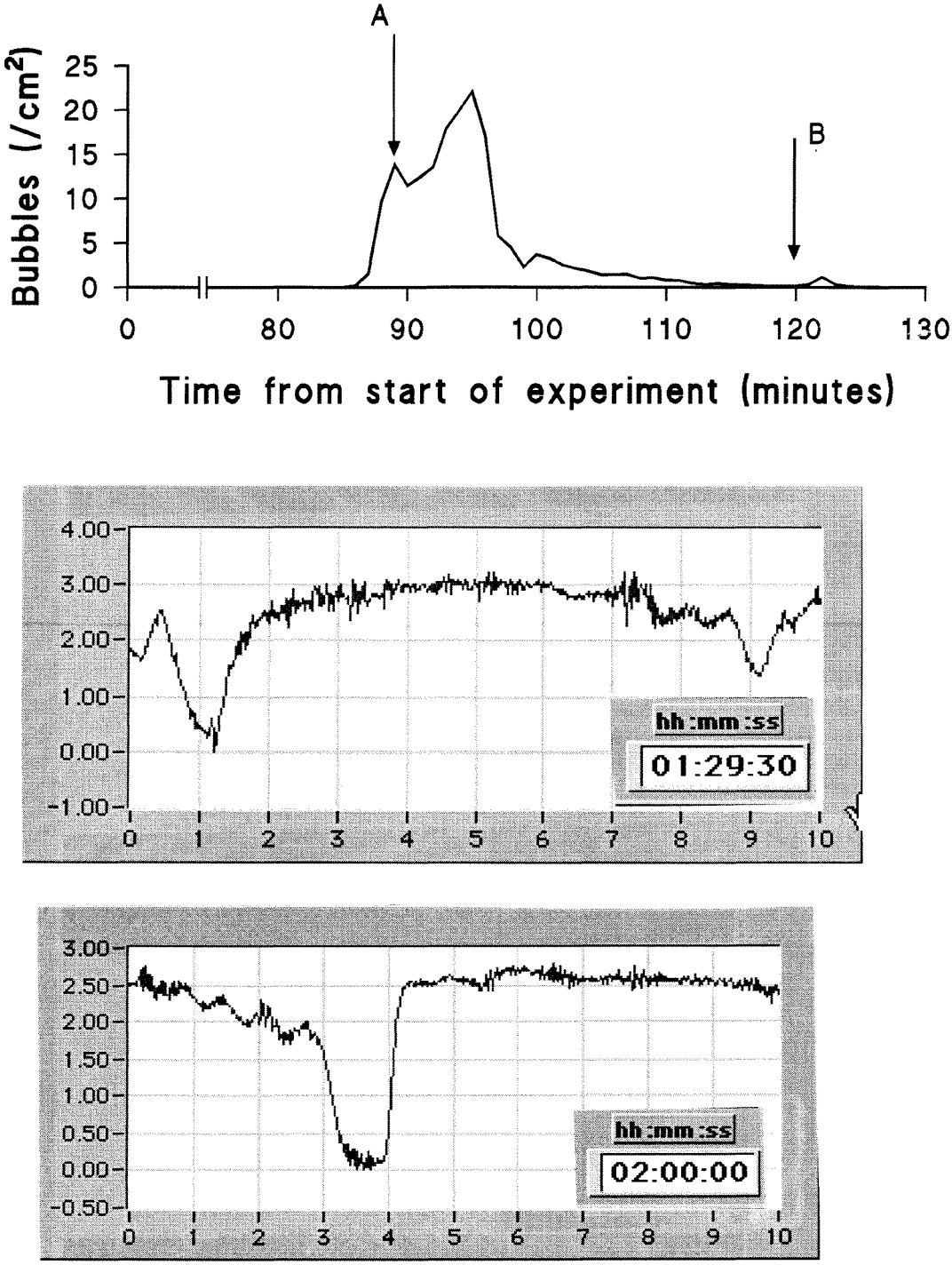


Figure 3 Respiratory patterns, experiment 2/4. See text for details

Apnoea is characteristic of herniation of the respiratory centre through the foramen magnum which is usually a consequence of increased intracranial pressure. The fact that a regular respiratory pattern was not restored after resolution of the bubbles supports this hypothesis, such herniation usually requiring intervention if it is to be reversed.

Theoretical calculations suggest that volume of the free gas phase in the brain is equivalent to a volume increase of 3 - 4%. The time course of the change in respiratory pattern is rather too quick to explain the initial change as resulting from anything other than a pressure increase due to the increase in volume as the free gas phase forms. However there is the possibility that the bubbles caused endothelium damage with resultant increase in extravascular fluids, leading to further increase in intracranial pressure over and above that caused by the increased free gas volume. Pou et al (1993) have shown that the exudation of lymph from the pulmonary vasculature continues after the resolution of gas bubbles; it is probably safe to assume a similar pattern in the brain. This would suggest that even though the free gas volume itself may not be enough to cause herniation the removal of free gas by treatment compression will not arrest progress towards the irreversible change, intracranial pressure continuing to increase after the removal of bubbles as fluid leaves the leaky vasculature.

The respiratory pattern seen in experiment 4 matched the patterns seen in the other animals which did not survive treatment.

CONCLUSION

The primary exposure used in this work was extreme and has apparently resulted in changes beyond those usually reported in the decompression treatment literature. In a severe decompression accident the focus of effort would normally be on initiating hyperbaric treatment as rapidly as possible. This work suggests that, in addition to working to reduce bubble formation and free gas volumes, attention should also be given to clinical procedures to reduce intracranial pressure. If so then the first stage must be to draw up guidelines to define the type of decompression accident which could cause a large enough free gas phase. In addition to the type of hyperbaric exposure used in this work free ascent, as used for submarine escape, is another possible situation in which increased intracranial pressure should be considered a likely cause of death. The same theoretical model as used in the current project also predicts the brain to be the main "target organ" for bubble formation in the submarine escape procedures (Flook 1997).

In decompression accidents of a severity similar to that described here the need to prevent increased intracranial pressure may shift the priority in the first 10 to 15 minutes away from hyperbaric treatment. Ideally both treatment to minimise the increase in intracranial pressure and the treatment to reduce bubbles would start without delay but in practice it may be necessary to consider holding off recompression for a few minutes to allow other treatment to be started.

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